AN ANALYTICAL STUDY OF
50 CASES ON CAUSES OF AMBLYOPIA

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CERTIFICATE

This is to certify that **Dr. R..Ramasubramanian**, MS. Post Graduate Student in Ophthalmology, Regional Institute of Ophthalmology, Government Ophthalmic Hospital, Madras Medical College, carried out this dissertation titled **An Analytical Study of Causes of Amblyopia** by herself under my guidance and direct supervision during the period of May 2004 to March 2007. This dissertation is submitted to Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the award of MS Degree (Ophthalmology).

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PART - I

INTRODUCTION

It is a condition with unilateral (or) bilateral decrease of visual function caused by form vision deprivation and (or) abnormal visual interaction, that cannot be explained by a disorder of ocular media (or) visual path ways itself. In appropriate cases it is reversible by therapeutic measures. It is caused by abnormal visual experience during early childhood, the critical period of development.

PREVALENCE:

It affects approximately 1-4% of the general population.

A study by Goel et al found that incidence of <1% in school children, the incidence was higher in rural school 0.7% than urban school 0.5% at primary level, probably because of lack of awareness among the rural population, about regular eye check up and the use of spectacles.

- The incidence of amblyopia in speciality clinics of strabismus and amblyopia ranges from 3-4% and 30-35%.
- It is a fairly common disease affecting 1% and 2% of the population in most developed countries.
- 2% of the population in most developed countries.
- 4.7% of patients with anisometropia had amblyopia (Deroirs from Netherlands) children with low birth weight less than <2,500 gms have twice the risk of getting amblyopia.
- Heredity also plays a role. In children born to amblyopic or strabismic arents the risk of amblyopia is 6 times higher.

CLASSIFICATION OF AMBLYOPIA:

1) Strabismic

2) Anisometropic (Unilateral (or) Asymmetrical)

    a) Aniso Hyperopic

    b) Anisomyopic

3) Form Vision Deprivation Amblyopia (Unilateral or Bilateral)

    a) Stimulus Deprivation amblyopia or pupil, media opacities (cornea, vitreous (or) lens) Unilateral occlusion or penalisation.

    b) Ametropic Amblyopia: Uncorrected bilateral high refractive error

        i) Hyperopia

        ii) Myopia

        iii) Astigmatism (Meridional Amblyopia)

4) Nystagmus related amblyopia

ORGANIC AMBLYOPIA:

    a) Subclinical macular damage

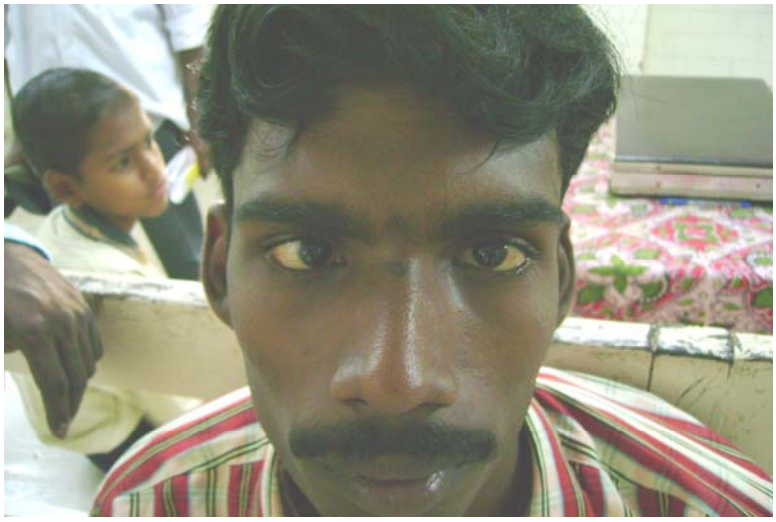
    b) Malorientation of cones

    c) cone deficiency syndrome

CASE OF ANISOMYOPIC AMBLYOPIA

A CASE OF CONGENITAL CATARACT CAUSING VISION DEPRIVATION AMBLYOPIA

A CASE OF STRABIS MIC AMBLYOPIA



CLINICAL FEATURES:

1) Decreased visual acuity (eg) Snellen's
2) Decreased grating acuity (eg) Tellers
3) Decreased Vernier acuity
4) Decreased (or) lost stereo acuity
5) Decreased contrast sensitivity
6) Decreased brightness perception
7) Increased perception and reaction time Naso temporal asymmetries in resolution of visual gratings.
8) Motility defects in pursuit, saccades and fixation.

VISUAL ACUITY DEFECTS IN DIFFERENT TYPES OF AMBLOYPIA:

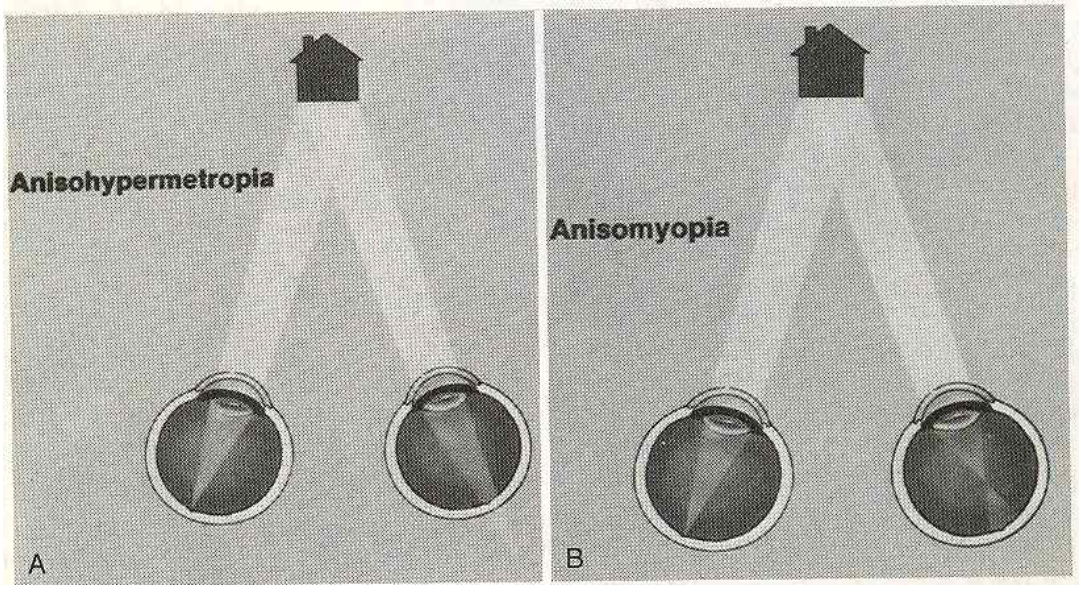
Recognition acuity is more affected than detection acuity recognition acuity is determined by Tellers, VER. Detection acuity is measured by catford Drum (or) Bailey Hall cereal test).

- Anisometropic and strabismic amblyopia behave diffently snellen's or Recognition acuity is more affected in strabismic amblyopic or mixed amblyopic compared to anisometropic amblyopia.

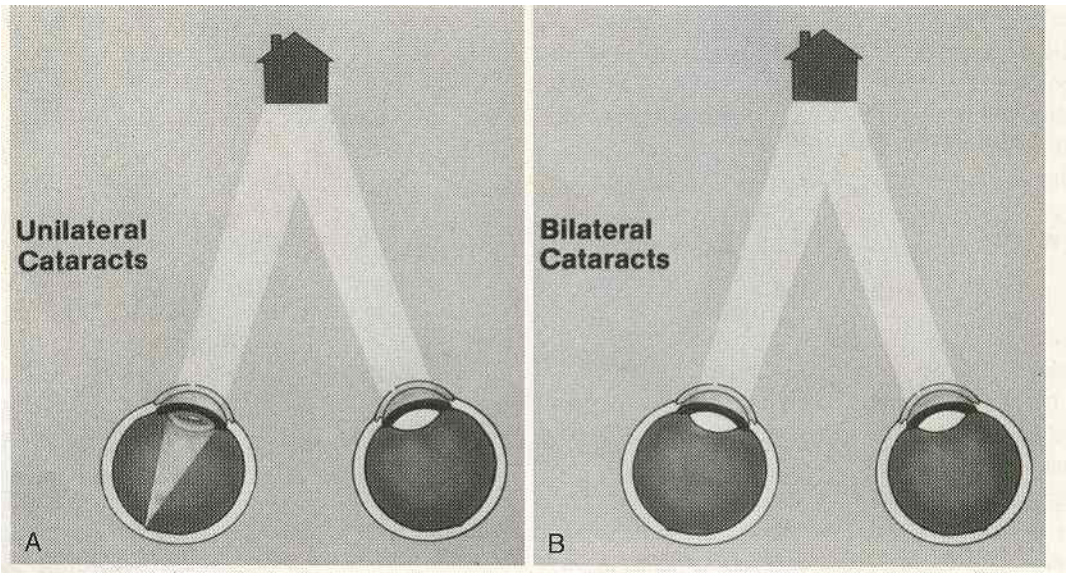
- Both snellens and grating acuity is affected equally in anisometropia amblyopic, whereas in strabismic ambloypes the grating acuity is affected to half of the extent of the snellen's acuity. Thus strabismic amblyopia is under estimated on grating test.

- For diagnosis of amblyopia any diminution of vision, difference between two eyes or in case of both eyes being affected, difference from the age related norm is taken to indicate amblyopia. Clinically a two line or snellen's chart (one octave difference) is considered significant.

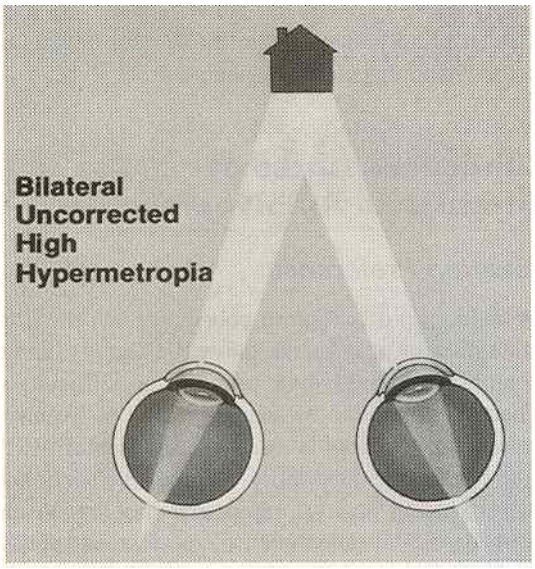
ANISOMETROPIA AND ANISOMYOPIA CAUSING THE RETINAL IMAGE IN THE MORE AMETROPIC TO BE OUT OF FOCUS



ONLY DIFFUSE AND REDUCED AMOUNTS OF LIGHT ENTER THE EYE THROUGH THE CATARACTOUS LENS OR BOTH THE LENSES



BLURRED RETINAL IMAGES IN BOTH THE EYES IN UNCORRECTED HIGH HYPERMETROPIA



TYPES OF HYPER ACUITY:

The visual apparatus is capable of making much finer spatial discriminations than the resolving capability of the retina may suggest (by Snellen's or grating acuity). this is called as Hyper acuity. Two common types of Hyper acuity are vernier acuity and stero acuity. Vernier acuity includes a variety of tasks that involve sensing the direction (or) spatial offset of a line or a point of reference. Vernier acuity can have an accuracy of 3-6 seconds of an arc or better. This processing is technically of sub pixel resolutions and is done at higher cortical areas. These are not easily influenced by retinal image motion or optical blur, implying there less likelihood of deterioration by uncorrected refractive errors or light media opacities. The later aspect has been used in predicting visual potential in the presence of a cataract.

Another well recognised feature of strabismic amblyopia is that vision is not degraded by neutral density filters, it may even show some improvement. However in anisometropic amblyopia an equal deterioration was seen. Other organic retinal pathologies causing diminution of vision are susceptible to deterioration by natural density filters. This distinguish functional amblyopia from these conditions.

ABNORMAL CONTOUR INTERACTIONS.

(CROWDING PHENOMENON)

Is seen in the form of degradation of visual acuity for objects placed in a row or line (Linear acuity), compared to the acuity of the same object viewed separately (single letter acuity). This phenomenon has been described as the crowding phenomenon).

Crowding phenomenon is present to some extent even in normal subjects (critical area of separation 1.9 to 3.8 min of arc) In amblyopia it is more pronounced similar to critical area of separation of peripheral retina in normal subjects (=8.4 to 23.3 min of arc). The crowding phenomenon has also been attributed to the poor visual acuity that is there in amblyopia.

The single letter visual acuity improves more rapidly during the course of treatment. Finally both the single letter and linear acuity should approach other, if it is not there is always a risk of recurrence of amblyopia.

PHARMACOLOGICAL EFFECTS ON VISION IN AMBLYOPIC EYES

P. Central norepinephrine System

Pottigrew and kasamatasu and kasomattu used the activation of central nor epinephrine system for the purpose of enhancing neuronal plasticity and kasamattu reported that catecholamine depletion prevented the ocular dominance shift seen in kittens after monocular occlusion.

NERVE GROWTH FACTOR:

Maffee and cocoorkers injected nerve growth factor into rats and found that exogenous NGF prevents the effect of deprivation, that is shrink age of cells in the lateral Geniculate Neurons and the function and anatomic organisation of the visual cortex. They speculated that loss of competition for the deprived eye is explained by the lack of neurotrophic factor. The monocular portion of the visual cortex of the deprived rats treated with nerve growth factor did not differ form the fellow eye.

LEVODOPA:

Administered as long as 1 week appears to provide positive results in the short term. The eye effects were explained in terms of the general role of dopamine both in the retina and in the visual pathway. Modest degrees of

improvement of visual acuity (2.7 lines) and contrast sensitivity (72%) have been reported which by were still significant 1 month after cessation of therapy. Legnoise and coworkers found an improvement which lasted 6 weeks after cessation of treatment with some side effects as nausea, head ache and mood changes.

Campos and coworkers reported a more permanent effect on visual acuity of the amblyopic and sound eye that lasted as long as 4 months.

CYTIDINE – 5 BIPHOSPHO CHOLINE (CITICOLINE)

Drug that increases the level of consciousness in patients who had trauma and parkinsonism. CITICOLINE improves membrane Adenosine triphosphatase (ATP ase) activity and modulates the turn over of catecholamines and serotonin. The age of the patients may be from 20-40 years, and thus beyond the age at which improvement can usually be expected because on improvement was found in both the eyes diplopia never occurred after treatment. Porciatti and coworkers showed citicoline in adult patients improves not only visual acuity but also contrast sensitivity and VER's. CAMPOS and coworkers evaluated the effect of citicoline in children with amblyopia. It was found that after 1 year follow up visuals acuity has improved more in patients treated with a combination of citicoline and part time occlusion that in those treated only with citicoline or with part time occlusion. It is noteworthy that no systemic side effects were found with this type of treatment.

LIGHT SENSE AND DARK ADAPTATION IN AMBLYOPIA

Both form vision and brightness perception in amblyopic are affected. Dark adaptation curves were essentially normal and even if there is an effect on the light sense. There is clearly a dissociation between the effect on the light sense and the acuity. While recovery time after a glare stimulus to fovea is normal, the perception time and reaction time is 6 times longer.

Pupils are generally normal and briskly reacting though afferent pupillary defect and raised edge light pupil cycle time has been reported by some workers.

It may be generalised that,

1) In amblyopia, the visual perception of fovea stimulates that of peripheral retina

2) The amblyopic visual system contains abnormally large receptive fields.

3) Functionally the amblyopic eye is at its best in mesopic and scotopic condition and worst at photopic condition.

BINOCULAR DEVELOPMENT AND AMBLYOPIA:

Though practical difficulties have limited the data pertaining to visual development in humans elegant and methodical experiments conducted by investigators on kitten and baby monkeys have revealed a lot. The conclusion were mentioned in human is by extrapolation from experimental work.

INTRA UTERINE DEVELOPMENT:

In the human retina most of the ganglion cells are generated between eight and fifteenth weeks of gestation, when the ganglion cell population reaches a plateau of 2.2 to 2.5 million. This is maintained till the thirtieth week when it starts to fall drastically due to rapid cell death for about 6-8 weeks. There after the process continues at a rapid rate through birth for the first few weeks of infancy. The ganglion cells at the final count are about 1 million. The loss of more than a million cells and their axons serve to fine tune the topography and specificity of the reticulo geniculate body in human is generated between gestational 8-11 weeks and by 10^{th} week the first retinal ganglion cells start invading the lateral geniculate body. The geniculate lamina emerge between 22-25 weeks. From an initial intermingling of inputs from each eye, segregation of & afferents occurs by pruning. The striate cortex cells appear by the age of 10-25 weeks of human foetus. The geniculate afferents begin to innervate the striate cortex by 26^{th} week. This has been demonstrated by injection of anatomic tracers. Initially geniculate afferents representing each eye overlap extensively in layer 4C. The segregation of

inputs into ocular dominance columns occurs during the last weeks of pregnancy and is almost complete at birth. Until shortly before birth there is a sort of loose wiring which is extensive and expensive, providing enough of material for fine tuning, which occurs by a method of making new usable synapses and breaking old (unused) synapses, synapto genesis.

In the retina two streams of ganglion cells Alpha (A cells) and Beta (B) cells are present. The A cells communicate via the magno cellular pathways, of LGB (Large cell Layers 1&2) to the cells of alpha of visual cortex area 17. The B cells of Retina communicate via the parvo cellular layer of LGB (Small layer 36) to 4C, Beta cells of the visual cortex, area 17.

PARALLEL VISUAL PATHWAY

Retinal glinglion cells	LGB	Cortex	Other
Alpha	Lamina	V_1 V_2	
		Thick stripes ↓	
	Magno cellular		V_3, V_5, M+, MST, V_4
Beta	Parvo cellular Inter strips blob thin stripes.		

Note:

V_1 striate cortex primary visual area (A_{17}) V_{2-} V_{3-} V_4 area in area 18, 19 and more anterior temporo parietal cortex.

The modular organisation of columns in the cortex is tuned to a variety of stimulus specifies such as accentuation, binocular disparity, motion perception etc. Each has discrete inputs and outputs. The Basic applied aspect of all of the development process is that the visual development is occurring by changes in the connections over a period of 0-5 years or more upto 9 years, during which is plastic until it reaches visual maturity later all these inter connections over a period of 0-5 yeras or more 9 years, during which is plastic until it reaches visual maturity. Later all these inter connections are difficult to be changed. The two patho mechanisms of different types of amblyopia are,

i) Form vision Deprivation and

ii) Abnormal binocular interaction

	Magnocellular	**Parvocellular**
I) Perception	Thick strips	9a) Inter blob. Inter strip
	motion	High spatial frequency acuity
	Flicker	some colour
	Transient	Blop thin strip
		colour
	Luminance	Low spatial frequency acuity (a) inter blob inter stripe
II Steropsis	40-80	6” – 1000”
	Local	Global
	Non cyclopean	Cyclopean
	Coarse	Fine
	Motion	Static
		b) Blob thin stripes depth tilt.
III. Oculomotor	Pursuit Vergence initiation	Vergence maintenance

THE ROLE OF VISUAL STIMULUS:

Visual experience loss of significant role to play in the afore mentioned synaptogenesis. In monkeys born by caesarian section with closed eyes. Precisely oriented simple and complex cells similar to the adult animals are seen, even with the orderly sequence of ocular dominance and orientation columns. Those are maintained in utero by spontaneous action potentials discharged by mammalian retinal ganglion cells. Abolition of these action potentials discharged by tetrodotoxin, prevent the normal prenetal segregation of retino geniculate axions into appropriate laminae of LGB. The same is observed after intra ocular administration of tetrodotoxin. If a newborn monkey is reared in dark (or) both eyes sutured, cells in the striate cortex develop bizarre receptive field properties, losing sharp orientation, tuning and normal bin ocular responses. After a prolonged period of deprivation, if the monkey is reintroduced into normal visual environment (lids are opened) the animal is profoundly blind with the minimal potential for recovery. These observations stress the role of visual stimulus for normal binocular development and are corroborated by good visual recovery only if early surgery and rehabilitation is done for bilateral or unilateral cataracts.

THE ROLE OF MONOCULAR DEPRIVATION:

The experiments by Hubie and Weisel has established the role of cortical competition in binocular visual development. To start with at birth all cortical cells have potential connection with both the eyes. If both eyes are functioning equally, the cortical cells driven by both the eyes re equal. About 10% of the cortical cells are driven by right eye and equal percentage by left eye. The rest 80% of the cells are driven binocularly C the central 20% of the equally by both eyes and the rest have a predominance of one eye or the other. If by any chance one of the eye is not functioning properly, the cortical cells of one eye is stolen or usurped by the other. This process of competition is easily reversible in the initial period of plasticity. Monocular deprivation produces a radical attenuation in the ocular dominance columns in striate cortex in favour the normal eye. It is believed that the two eyes compete for synaptic contacts in layer 4c of visual cortex. monocular eye lid closure imposes a severe handicap in this contest. as a consequence of the deprived eye loses many of the connections already found at birth. The ocular dominance columns of the deprived eye shrink and those of the favoured eye swell. a similar changes is observed in the laminae of the LGB., although, there is no competition at this level. the LGB cells of deprived eye are smaller as they are required to sustain a lesser arbor of axons in layer 4c of cortex.

IMPORTANT CONCLUSIONS:

Some important conclusions from these studies are

i) The loss of binocularly innervated striate neurons is not specific for ambyopia, it occurs after any brief disruption of binocular input in early life. this has been correlated to stereopsis.

ii) The decrease of cells responding to the stimulation of the amblyopic eye is highly specific for amblyopia, regardless of the etiology and correlated quantitatively with the decrease of visual activity.

iii) Even one week of disruption is sufficient to cause amblyopia in the sensitive period. occlusion amblyopia develops rapidly in children upto 4 years but can occur later also.

iv) Recovery of neurons connected with the amblyopic eye doesnot occur on treatment, but improvement of binocular cells appears to be permanent at least in monkeys, after binocular vision early in life. infantile esotropia of early onset doesnot allow good stereopsis.

v) In anisometropic and vision deprivation amblyopia both monocularly and binocularly innervated portions shrink, but in strabismic amblyopia only the binocularly innervated part shrinks in LGB. In the cortex, the same is observed for vision deprivation amblyopia, but need to be confirmed for anisometropic amblyopia.

vi) Nerve growth factor (neurotropin) seems to prevent shrinkage in LGB in rats in strabismic and vision deprivation amblyopia yet to be confirmed in primates.

THE APPLIED ASPECT IN CLINICAL PRACTICE:

The same process occurs during occlusion therapy. In the initial period of therapy, the vision improves in the amblyopic eye, by the take over of the other selected cells of the other eye and is reflected in the drop in the visual acuity of the normal eye. However gradually the earlier “unselected” cells are recruited for the amblyopic eye. This all the gain of the amblyopic eye is not due to loss of good eye. But loss is possible if the normal eye is not given chance intermittently a breather. Then it results in occlusion amblyopia which is a type of vision deprivation amblyopia and has grave prognosis.

THE CRITICAL PERIOD

The critical period corresponds to the fine phase, when the wiring is still malleable. It should be clear that amblyopia is likely to be corrected, occlusion amblyopia is also possible. It has been observed to differ for different types of amblyopia. For vision deprivation amblyopia the upper limit is 6 years and for anisometropic amblyopia it is 8 years. The latter cases do respond even in the teenage, whereas the strabismic amblyopes do not respond after 12 years.

VISUAL ACUITY ASSESSMENT

TYPES OF VISUAL ACUITY

1. Minimum visible (which is actually a function of brightness and is about 1 second of arc).

2. Minimum separable (which hyper acuity, a function of higher cortical centres and is about 16 – 20 seconds of arc more, than the maximum potential of foveal cones therefore called as hyper acuity vernier and stereo acuity are examples).

3. Minimum resolvable and minimum recognisable (which is what is understood by visual acuity and is limited by foveal configuration). the finest acuity can be of 30 seconds of arc.

TESTS FOR VISUAL ACUITY

The tests for visual acuity can be grouped into – three types

a. Detection acuity test:

They assess the ability to detect the smallest stimulus (without recognising correctly) eg

1. Catford drum

2. STYCAR graded balls test

3. Schwarting’s metronome

4. Boecks candy beads

5. Dot visual acuity

b. Recognition acuity test:

They assess the ability to recognise the stimulus (or) distinguish it from other competing stimuli.

a. direct identification test

- Sjogren hand test
- Landolt C test
- Snellen’s E
- Arrows

b. Letter identification chart

- Snellen’s chart
- Sheridan letter test (STYCAR)
- Lippmann’s HOTV test

c. Picture identification charts / miniature toys

- Beale collin’s picture charts
- Light house test
- Allen’s picture cards
- Domino cards test
- Miniature toy test of sheridan

d. Picture identification on behavioural pattern

- Bailey hall cereal test
- Cardiff acuity cards
- OKNOVIS based on arresto – visuography.

3. resolution acuity test:

- Opto kinetic drum
- Preferential looking tests – forced choice or operant type teller acuity cards.
- Visual evoked responses.

VISION TESTS IN INFANCY:

CATFORD DRUM TEST:

It was devised CATFORD and OLIVER useful in infants and pre school children. It is based on observation of pendular eye movement (oscillatory eye movement not an opto kinetic nystagmus). That is elicited as the child follows an oscillating drum with dots. The dots are displayed in various sizes (15mm to 0.5mm) and the test distance is 60cm (2 feet). The dots represent 20/600 to 20/20 vision. The smallest dot that evokes the pendular eye movement determines the visual acuity. Unfortunately it is known to over estimate vision by double or quadruple times and is unreliable for amblyopic screening.

CARDIFF ACUITY CARDS

They are based o the principle of preferential fixation on cards which have a picture optotype and a blank located vertically. The picture upto types are of the same size, but have been especially drawn with two dark lines with a white space of varying width, in between, such that the picture is visible only at a particular distance or closer. These are known as vanishing optotypes as they vanish at a farther distance. They have been developed at cardiff UK. The child can identify the picture by verbalising, pointing or fixation preference. They are more likely to be in Recognition acuity group. The visual acuity is described in Snellen’s notation.

VISUAL EVOKED RESPONSE (VER)

VER records the change in the cortical electrical pattern detected by surface electrodes monitoring the occipital cortex following light stimulation of the retina. The stimuli may be a pattern checker board or stripes on an unpatterned flash. The size of grating or checks is to 30,15 minutes of arc. Layer check sixes than 60 minutes (1^0) are fallacious for visual acuity. The P_{100} amplitude and latency is noted. If the Latency is not between 100-145 M sec, it cannot be relied upon and should indicate poor vision. The VER can be recorded in two modes: Transient and steady state.

TRANSIENT VER. Where abrupt unique alternation of stimulus is at a relatively low rate so that each stimulus generates a separate VER output.

STEADY STATE VER.

Where rapid rate of stimulation causes blending of output into a continuos wave.

Visual acuity of 6/12, 6/6 have been estimated at 6 month of age by VER. These estimates of visual acuity have not been very reliable and are lighter than by other methods. They may reflect the electro physiological changes but not truly indicate the visual characteristics which is a perceptual phenomenon. They are however useful in giving an objective record of the underlying visual pathway and to exclude organic pathology.

INDIRECT ASSESSMENT OF VISUAL ACUITY:

i) **Reflex responses:** The pupil reacts to light after 29 week of gestation. The baby begins to turn its head to diffuse light after 32^{nd} – 36^{th} week of gestation.

ii) **Fixation:** The fixation reflex is a prerequisite for normal visual development and is present at birth. Even in preterm babies after 33 weeks of gestation. But it may not be well established. 75% of infants fixate by two weeks and 100% by two months. New borns show fixation preference for moving stimuli, blinking light, patterned stimulus, stimuli with high contrast stimuli with especially red green and human face. Near objects are preferred. The fixation may not be steady and is interrupted with refixation. The spans of fixation lasts from seconds to minutes.

Follow Movements

Following horizontally moving targets has been seen in full term new borns and well developed by first month. Vertical tracking usually is elicited by 4-8 weeks. The movements may have jerky interruption and corrective movements are slow. The range of following is 45^0 at birth, 90^0 by 4 weeks and 180^0 by 3 months.

FACTORS IN MATURATION OF VISION:

i) Though rods and cones are distinguished even 15 weeks prior to birth they do not attain adult like dimensions until 14 months after birth.

ii) Myelination of visual pathways is completed by one month, but the amount of myelin increase in subsequent months upto 2 years.

iii) Cortical neuronal dendritic growth and synapse formation at 25 weeks of gestational age and is very active in the first two years.

These factors correlate with vision as well as other functions like

i) Accommodation which is minimal at 1 month of age, but well developed by 4 months of age.

ii) Fusion, starts of 2 months of age and fusional convergence develops by 6 months.

iii) Stereopsis is present at 1-2 months. Well developed by 4 months, though adult levels may be seen at 5-7 years of age.

B) Vision testing in 1-2 yeras.

Boeck candy test, worth's ivory ball test, sheridan's ball test.

C) Visual acuity in 2-3 years.

1) Miniature toys test: Pairs of miniature toys are used. The child is asked to name or pick the paid from an assortment. Test distance is 10 feet.

Central steady fixation: Usually means good potential for vision. In new borns atleast 6/60. Later this should indicate 6/9 – 6/6 vision.

A preference of fixation with one eye over the other indicates poor vision in the non preferred eye. Resentment of the closure in one eye indicates poor vision in the other eye.

Induced Prism test:

Assessment of visual potential on the preference of fixation of one eye, over the other is simple in the presence of a squint. In the absence of squint a deviation can be induced by introducing a 10-15 pd prism base out, the child is forced to choose to fixate with one eye or the other.

FIXATION AND VISION:

Fixation pattern	Visual Acuity
Gross Eccentric fixation	Less than CF at 1 metre
Unsteady central fixation	Less than 6/60
Steady central fixation but not maintained	6/60 – 6/30
Steady central fixation can maintain but prefers other eye.	6/24 – 6/18
Central steady fixation free alternation or cross fixation	6/9 – 6/6

AGE OF CHILD AND VISUAL ACUITY	
Age in months	**Visual Acuity**
1	6/120
2	6/60
12-18	6/48 – 6/12
18-24	6/24-6/7.5
24-30	6/15-6/7.5
30-36	6/12-6/6

Coin test:

Coins of different sizes at different distance are shown. The child is asked to distinguish between the two faces of the coin.

Dot Visual Acuity Test:

In a darkened room the child is shows an illuminated box, with presented black dots of different diameters, one at time, successively smaller dots are shown. The smallest dot identified correctly twice is taken as acuity threshold.

VISION TESTS IN 3-5 YEARS:

i) Tumbling E test:

The test is preferred vision test for mass screening in pre school children. It consists of different sizes at E in one of the four positions right, left, up or down. After familiarising the child, he indicates the direction in which 'E' is oriented. Which he does by hand or orally. It is done at 6 metre distance and each eye is test separately. Single letter acuity is supposed to be better in amblyopes in comparison to line acuity on chart due to crowding phenomenon 'E' charts with surrounds have been suggested to offset this disadvantage.

ii) Landolts C with is used in a similar manner as also Sjogren's hands and arrows.

iii) Sheridan letter test. The sheridan test uses 5 letters H.O.T.V. and in the fine letter test, A and U are added in 7 letter set. C & L are also added in 9 letter set. Testing distance is 10 feet (3 meters). The child is expected to name the letters (or) indicate similar letter on the card in hand.

iv) Lippmanns HOTV test: The method is simpler version of Sheridan's test using only 4 letters H, O, T.V at a distance at 3 meters. The method is the same as above.

TESTING OF HYPER ACUITY:

VERNIER ACUITY TESTING:

Vernier acuity tests are of interest particularly in amblyope, whereas grating acuity may be fallaciously normal. In has been used in the acuity card format for infants and small children using the AFC pattern.

Steroacuity:

Has been seen to be developed and testable on the PLT by 6 months. It has been used as a screening test for binocular vision anomalies in pre school children but with difficulty.

Steroacuity test:

The real depth tests are not used, most clinical tests are based on haploscopic principle using two dimensional or vectographic pictures. Some elments of the two pictures have a disparity which is fused to create a 3D image.

This can be test on

i) TNO test with red green goggles

ii) Synoptophore; with stereopsis slides

iii) Randot Stereo test and titmus stereotest with polaroid spectacles.

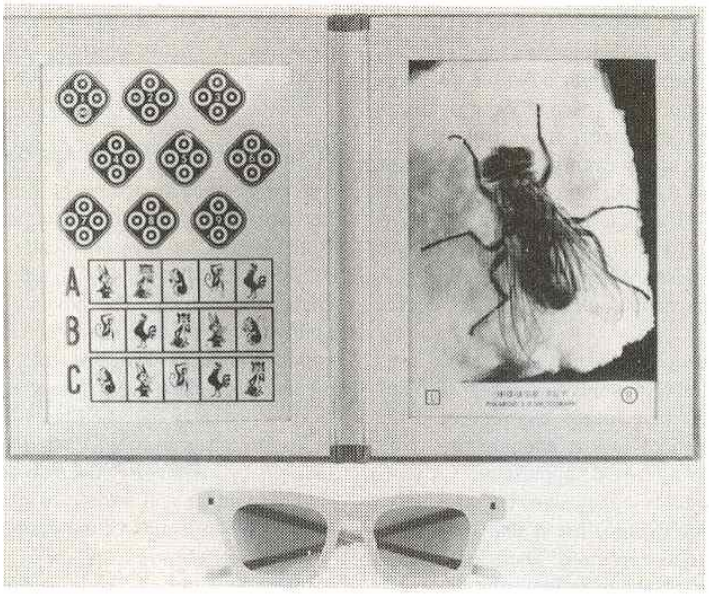
iv) Lang acuity test using no glasses.

v) Special 3D pictures.

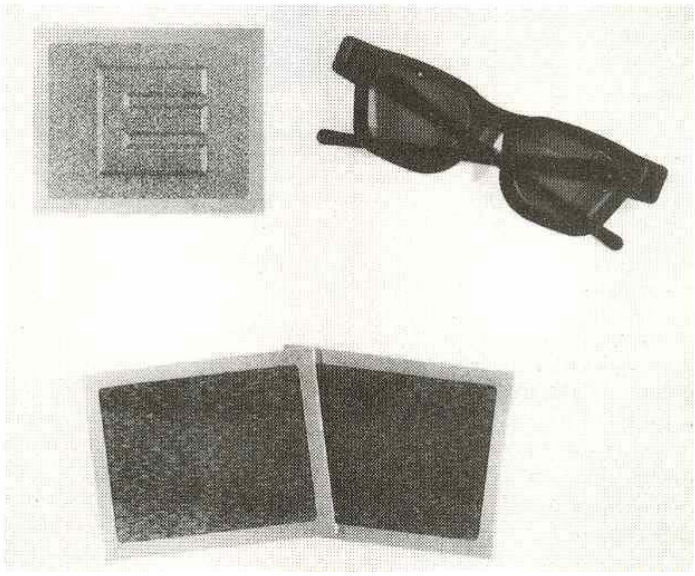
SYNOPTOPHORE TESTING

This is used to test the binocular single vision and angle of Deviation

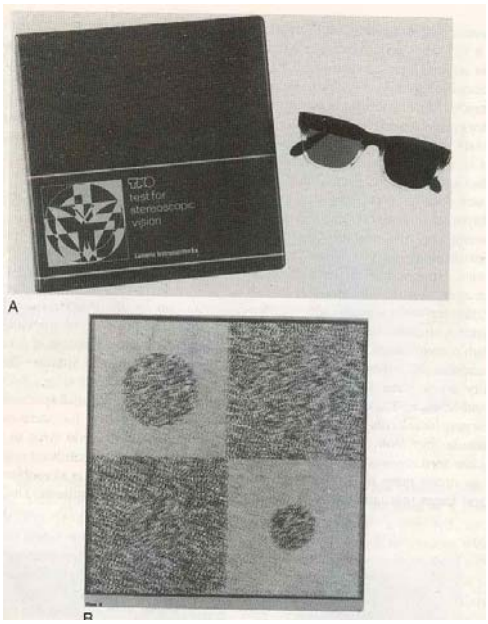
THE TITMUS STEREO TEST



RANDOT E - TEST



A THE TNO TEST . B. THE RANDOM DOT STEREOGRAM



The last two examples in which the dissociation is not achieved by glasses, which may not liked by children.

The Langs test is based on the principle of panography when two images are printed on the same card each interrupting the other with regular linear interruptions. A prismatic film laminated over the picture ensures that one image is visible to the right eye only and the other to the left eye only. The two when fused in spite of the disparity create a 3D vision.

RANDOT STEREO TEST:

This is the most popular clinical and has replaced the earlier popular titmus fly test. It uses Julesz's Random dot back ground to mask the monocular cues which are there with the animal test and wirts circle test. Geometic figures like square, circle, triangle, star etc. are also presented. The latter type figures, though a better test are usually not appreciated by small children. The test requires polaroid glasses to be worn by the patient. It is used at a distance of 40cm and thus test near binocular vision, so that myopes upto 3 diopteres can be missed in a screening test. The wirt's circle 1-40 test the streo acuity from 400 arc second to 20 arc seconds.

TNO test:

This test is also based on the random dot back ground but uses red green glasses for dissociation of the two images. It tests steroacuity from 480 are seconds to 15 are seconds.

FRISBY TEST:

The Frisby Stereotest consists of three Perspex plates of differing thickness 6mm, 3mm and 15mm on one face of each plate are found squares. Three of which are filled with a random pattern of blue triangles of various sizes and fourth of which has a central, circular area that is not patterned. On the opposite side of the plate coincident with this area is a circular pattern of similar blue triangles. The plate is held in a white board and when viewed directly, the squares are all filled with random patterns although in one square a binocular viewer will see a circle standing up from the plate C crossed disparity or lying below the rest of the design (uncrossed disparity) depending on which side of the plate and distance from the subject different stereo acuities can be assessed. For 30cm viewing distance, the 6mm, 3mm and 1.5mm represent 600, 300 and 150 are seconds of stereo acuity respectively. This assumes the inter pupillary distance of 60mm, but not significant change is caused by different IPD.

DISTANCE STEREOPSIS TEST

Stereo acuity should be tested for distance also. A projection vectographic or oculus distance stereo tests can be used to test stereopsis for distance. A diministed stereopsis for distance may be on early sign of decompensating exophoria.

NORMAL STEREO ACUITY:

Though adult individual are capable of appreciating stereopsis with disparties as fine as 15-20 arc seconds. The adult norm is 40 arc second. For children 3-5 year old the norm is 70 arc seconds, and for 5-7 years it is 50 arc seconds. Children above 8 year have the adult no norm.

GROSS STEREOPSIS:

In the absence of fine stereotests a gross estimation of stereopsis can be made by a bed side test. Two pencil test was popularised by Lang. A pencil is held in the examiners hand horizontally and the child is asked to touch the tip with the tip of another pencil rapidly come from one side. Care should be taken to avoid giving the end on view of the pencil, as that can be accomplished even monocularly, therefore horizontal pencils are better as they do not allow an end on view. Always compare the binocular task. The test is a gross stereopsis test of about 400 arc seconds disparity. A rough estimation of visual acuity has been made on the basis of stereo acuity.

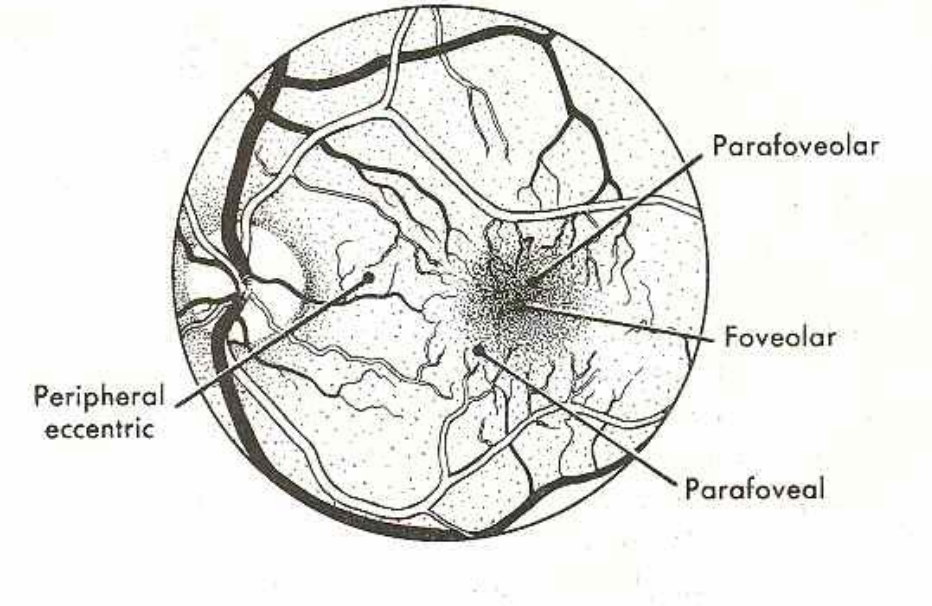
LANG'S TWO PENCIL TEST

A pencil is slide in the examiner hand and the child is asked to touch the tip with the tip of another pencil rapidly from one side

TYPE OF FIXATION

Site and type of fixation whether it is steady or unsteady and whether it is foveal (within 2^0) parafoveal (2^0-5^0), para macular (5^0-10^0), paracecal or peripheral (or) temporal. A steady central fixation is a good prognostic sign. An unsteady but central foveal fixation indicates a possibility of good vision with conventional occlusion. But a steady peripheral or para macular or paracaceal fixation generally indicates a poor prognosis. Eccentric fixation is generally unilateral with the other eye showing central fixation. But some times one may encountaer bilateral eccentric fixation. This is usually vertical ie the fixating points are above or below the foveal in both the eyes. It indicates a macular pathology which may be missed clinically. Electro physiological tests:- EOG and ERG done is such cases confirm and prognosticate such cases.

VARIOUS TYPES OF NONFOVEOLAR FIXATION



TREATMENT OF AMBLYOPIA

1) Amelioration of the amblyopiogenic factor
2) Occlusion
3) Penalisation
4) Pleoptical
5) CAM vision stimulation
6) Red filter treatment
7) Medical treatment
8) Active vision therapy

1) Amelioration of amblyopiogenic factors

Removal of amblyopio genic factor is an essential pre requisite followed by proper visual rehabilitation for distance and near vision. Needless to say that a uniocular or bilateral congenital contract or traumatic cataract or traumatic cataract to be given both distance and near correction.

2) Occlusion:

Since 1743, when de Buffon first described occlusion has remained the sheet anchor therapy for amblyopia. In this therapy the amblyopic eye is given a preferential chance of development, as the dominant eye is totally withheld from binocular participation. A properly done occlusion with good compliance ensures an almost 100% success rate, especially if treated upto 7 years of age. The success rate recedes with increasing age.

TYPES OF OCCLUSION:

a) Total and b) Partial

Total Occlusion:

Which completely obscures both light and form vision and is the type usually advocated for moderate to severe amblyopia.

i) Direct skin patch A cotton eye pad patched to the eye with the help of a micropore plaster.

ii) Spectacle patch.

iii) Doyne's occluder: A black rubber occuder which sticks on the back of the spectacle glass by suction.

iv) Pirate patch

v) Contact lenses

PARTIAL OCCLUSION

This degrades the vision of the normal eye so that the amblyopic eye has an advantage. this is a milder form of penalisaiton and is used for milder amblyopia or in recovered cases for maintenance of binocular vision. It also requires correction of factors like squint anisometropia or anisokeinia suitably. The advantage over total occlusion of this modality is that it offers binocular stimulation. Layers of transparent scratch tape or colourless nail varnish can be applied on the back surface of the dominant eye.

Another way of differentiation of period of occlusion,

a) Full time occlusion: All waking hours virtually 24 hours.

b) Part time occlusion this is for gradual duration, different waking hours of the day on the basis of the age of the child,

Duration of occlusion

Full time occlusion is advocated by

It is advised for 24 hours (full day) for,

2 days for 2 years old

3 days for 3 years old

4 days for 4 years old

5 days for 5 years old

6 days for 6 years old

This is alternated with one day of occluding the amblyopic eye when the dominant eye is opened occlusion of the dominant eye is called conventional occlusion and occlusion of the amblyopic age is called inverse occlusion. The follow up period for occlusion is 3 months. There is a risk of occlusion amblyopia in dominant eye during occlusion, to avoid this the child has to be reviewed every 15 days.

PENALISATION

Penalisation has the advantage of being cosmetically acceptable, but it doesnot inhibit the abnormal binocular interaction which is the essential cause of amblyopic. Its indications are limited (eg) moderate amblyopia, in uncooperative patient, anisometropic amblyopia and as maintenance therapy. The major disadvantages of penalisation include active inhibition is not eliminated, risk of occlusion amblyopia persists and also the cost of drugs.

CAM Stimulator:

Campbell and coworkers proposed a new treatment for amblyopia. The discs are made with light and dark bars of various widths and rotated at the rate of one rotation per minute to provide different orientation to stimulate a variety of brain cells. Seven discs of various spatial frequencies are used. Patients views

them for seven minutes. This is based on the principle that the visual areas of brain respond to stimuli of grating of a specific size at a certain orientation. This modality is not used now a day.

Pharmacological Therapy for Amblyopia:

L - Dopa:

Various studies indicate the improvement in vision of amblyopia after L-Dopa therapy.

Gottlob et al shows that L-Dopa improve contrast sensitivity and reduces binocular suppression in the affected eyes of adult human anisometropic and strabismic amblyopes. Gott lob et al further investigated the effect of Levo Dopa with week daily administration of using a cross over double masked design. Visual acuity improved in 70% of patient after one week of administration of Levo-Dopa and the improvement of visual acuity and visual fields persisted after completion of Levo Dopa administration.

Citicholine (Cytidine 5-Diphospho Choline)

Citicholine administration in adult volunteers (1 gram / day 1 / for 15 days) of strabismus. Amblyopic demonstrated improvement lasting over 6 months in visual acuity of both amblyopic and dominant eyes. Even contrast sensitivity and VEP were significantly improved.

PART - II

PART – TWO

AIMS AND OBJECTIVES

- To find out the relative proportions of different types of ambloypia in a referral centre.

- To study the characteristics associated with different types of amblyopia in our population the extent of visual impairment produced by different types of amblyopia at presentation.

METHODS AND MATERIALS

Over a period of two years 2004 October to 2006 September, 1800 patients were see in strabismus and Paediatric Ophthalmology clinic at RIOGDH, Chennai. This is a prospective study of 50 cases of amblyopia.

INCLUSION CRITERIA:

1) Patients aged between 6 months and 60 years of age.

2) No previous history of strabismus surgery.

3) All patients with corrected or uncorrected refractive errors

4) All patients referred from Institute of child health for ophthal evaluation to squint clinic.

DEFINITION CRITERIA:

1) A difference in the best corrected visual acuity between the two eye in the Snellen's line equivalent measure on the Teller's acuity in children less than 4 years in absence of any organic lesion that could result in decrease of vision.

2) A BCVA of less than 6/12 bilaterally on the Snellen's chart / equivalent measure on the teller's acuity chart in children less than 4 years in the absence of any organic lesion that could result in decrease of vision.

The assessment included, history of

1) Age of onset as noticed by patients or gaurdians.
2) H/O Low birth weight
3) Family history of strabismus
4) Patient coming from Rural or Urban areas.

General and systemic examination were done to rule out any associate problems.

Ocular examination included.

1) Unaided visual acuity
2) Best corrected spectacle visual acuity

   (With the help of snellen's chart in patients above 4 years and Teller's acuity chart in children less than 4 years of age.

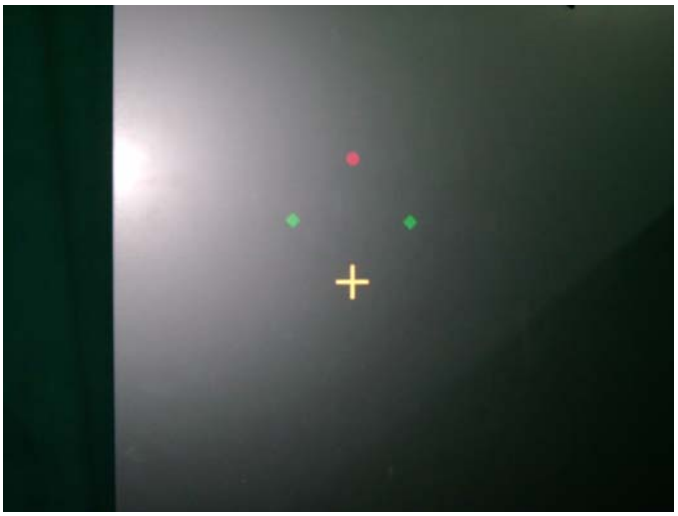
3) In infants fixation and preferential looking patterns were noted.
4) Refraction was made under appropriate cycloplegics according to the age of the patient, assessment of ocular alignment and motility and associated strabismus.
5) Slit lamp examination is done to rule out any anterior segment pathology.

6) A detailed fundus examination is done to rule out any posterior segment pathology.

7) Assessment of binocular status of the eye is done with the help of Worth Four dot test.

WORTH FOUR DOT TEST

The images of two eyes are dissociated using red green glasses.



The patients with the normal binocular single vision sees two green dots, one red dot and one plus.

STANDARD DEFINITION CRITERIA FOR DIFFERENT TYPES OF AMBLYOPIA

1) STRABISMIC AMBLYOPIA:

Defined as amblyopia in the presence of Heterophoria for distance and near fixation. Patients with strabismus along with refractive errors of more than 1 D spherical equivalent in one or both eyes or eyes with regular astigmatism, 1.5 D cylinder astigmatism in any meridian were included.

2) ANISOMETROPIC AMBLYOPIA:

This included patients who had amblyopia in the presence of anisometropia. That was 1-D or greater in spherical equivalent or a1.5D or greater difference in astigmatism in both the eyes that persisted for at least 4 weeks after spectacle correction in the absence of any measurable heterotropia at distance or near.

3) SENSORY DEPRIVATION AMBLYOPIA:

This group included patients with known documented cause of sensory deprivation with no primary heterotropia or refractive errors, that could be causally related to the amblyopia.

4) AMETROPIC AMBLYOPIA

Patients with refractive errors more than 1D spherical equivalent in both the eyes or one eye and no associated strabismus or any other ocular pathology were classified under this category. Patients with significant anisometropia (as defined above) along with high refractive errors in both the eyes were excluded from this category and were classified under strabismic amblyopia.

5) MERIDIONAL AMBLYOPIA:

Patients with regular astigmatism 1.5D of astigmatism in any meridian or those with irregular astigmatism or both eyes, resulting in a decrease in vision in one or both the eyes and no associated strabismus were classified as having meridional amblyopia. Patients with significant anisometropia (as defined above) along with the difference of 1.5D or greater astigmatism between the two eyes were excluded from this group and included under anisometropic amblyopia. Patients with heterophoria for distance and near with regular astignatism more than 1.5D in any meridian or irregular astigmatism were included under strabismic amblyopia.

OBSERVATION AND RESULTS

AGE AND SEX DISTRIBUTION

Age Group	No. of patients
< 2 years	5
2 – 5 years	8
5 – 10 years	23
10 – 20	10
> 20 years	4

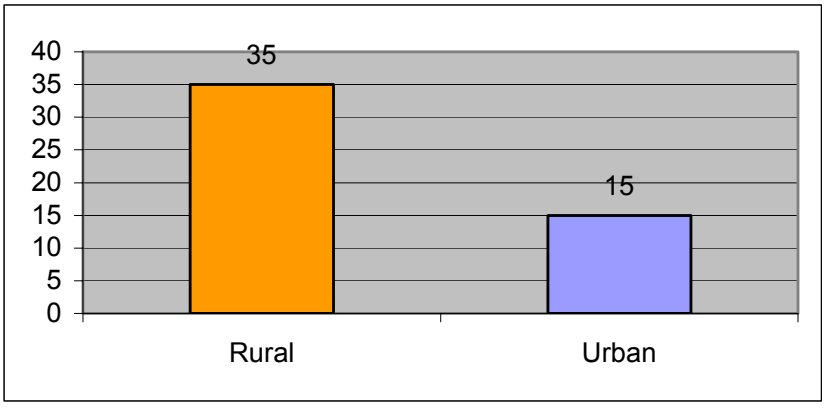
SEX DISTRIBUTION

Sex	No. of Patients
Male	30
Female	20

Average age of presentation of amblyopia in our study is 10 years.

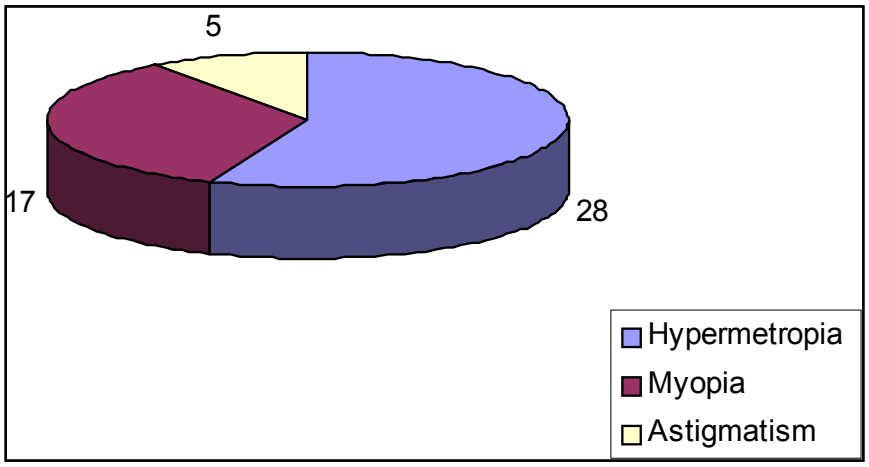
RURAL AND URBAN PROFILE

NO. OF PATIENTS	
Rural	35
Urban	15



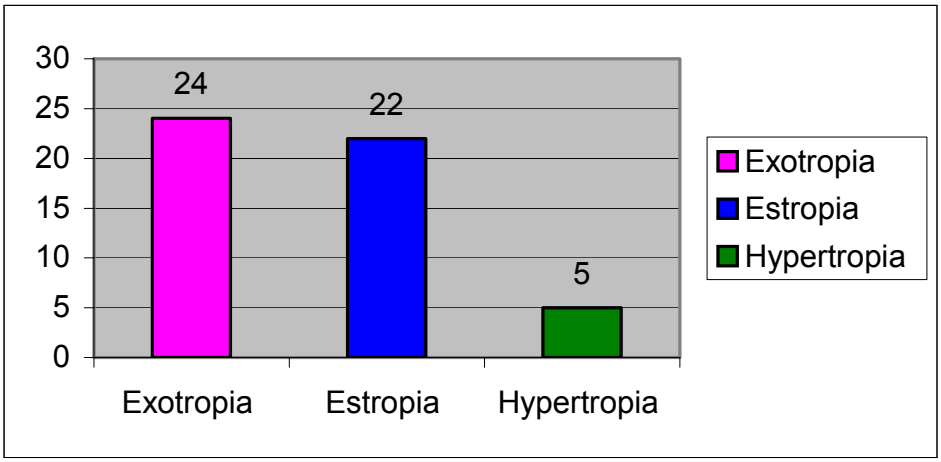
INICIDENCE OF DIFFERENT TYPES OF REFRACTIVE ERRORS IN AMBLYOPIA

NO. OF PATIENTS		
Hypermetropia	28	56%
Myopia	17	34%
Astigmatism	5	10%



INCIDENCE OF DIFFERENT TYPES OF STRABISMUS

NO. OF PATIENTS		
Exotropia	24	48%
Estropia	22	44%
Hypertropia	5	10%



PERCENTAGES OF VARIOUS TYPES OF AMBLYOPIA

Type of Amblyopia	No. of Patients	Percentage
Strabismic	38	76%
Ametropic	4	8%
Anisometropic	3	6%
Meridional	1	2%
Vision Deprivation	4	8%

COMPARISON OF TYPES OF FIXATION IN STRABISMIC AMBLYOPIA AND MERIDIONAL, ANISOMETROPIC AND AMETROPIC AMBLYOPIA

	Strabismic	Ametropic, Anisometropic and Meriditional
Eccentric	8 (21.05%)	3 (37.5%)
Perifoveal	6 (15.7%)	3 (37.5%)
Parafoveal	18 (47.36)	2 (25%)
Peripheral	6 (15.7%)	0

PERCENTAGE OF PATIENTS HAVING LOW VISION <6/60

No. of Patients		
Strabismic	17	54%
Anisometropic, Ametropic, Meridional	4	33%

- 4 patients in our study had Nystagmus 8%.
- 6 patients had family history of strabismus 5 patients had history of low birth weight
- One patient had Duane's Retraction Syndrome
- One patient was diagnosed to have chronic progressive external ophthalmoplegia

DISCUSSION

Amblyopia is one of the most common causes of visual impairment in both children and adults. It affects 4-5% of general population and its incidence in paediatric ophthalmology and squint clinic is 30-35%.

AGE PROFILE

The average age of presentation is 10 year of age which is very late to start treatment. This is due to lack of knowledge in the general population and the ignorance of yearly eye check up in children, this can be brought down by better awareness among the general population and screening programmes.

ADULT AMBLYOPIA:

The percentage of patient presenting after 10 year is 28% (14 patients). The relative proportions of different types of amblyopia were strabismic 76%, Ametropic 8% visual deprivation amblyopia 8%, meridional 2%, and anisometropic amblyopia 6%. This proportions were different from General population since many of patients were referred from secondary referral centres with strabismus. And because of the inclusion creteria used here the relative proportions of different types of amblyopias is different and the selection bias as it is a hospital based study and is not representative of the population.

Fixation Pattern:

Patients stratismic amblyopia had low percentage of perifoveal fixation 15.7% as compared to other types of amblyopias 37.5% clumped together.

Type of Amblyopia	Our study n=50	Study Conducted by Menon Vimla, Chaudhuri Zia, et al at AIIMS
Strabismic	76% (38)	37.38
Anisometropic	6% (3)	31.1
Ametropic	8% (4)	18.31
Meridional	2% (1)	5.56
Vision Deprivation	8% (4)	7.63

In the incidence of refractive errors hypermetropia has highest incidence of 56% followed by myopia and Astigmatism.

21 patients had a visual acuity of 6/60 and below and this accounts to 42%.

Rural, urban profile:

This study showed a relatively higher percentage of rural population 70% compared to urban population 30%. This is because of the lack of awareness among the rural population.

CONCLUSION

- The prevalence of amblyopia in our squint and neruophthalmology clinic was 30-35%. Amblyopia is a very common cause of impaired vision in child hood.
- The incidence of anisometropic and ametropic amblyopia is compared to be less in our population compared to western countries.
- 94% of the patient in our study had squint.
- Hypermetropia is the most common refractive error associated with the amblyopia followed by myopia and astigmatism .
- Esotropia is the most common form of strabismus associated with the amblyopia.
- 10% of patients with amblyopia had low birth weight.
- 12% of patients had family history of strabismus.
- The relative proportion of rural population compared to urban population is higher.
- 8% of patients with amblyopia in our study had Nystagmus.
- 42% of patients in our study had low vision C 6/60.

Earlier diagnosis and health education and screening programmes in rural areas can reduce the prevalence of amblypias.

PROFORMA

EVALUATION OF AMBLYOPIA

NAME AGE SEX

Address:

Presenting Complaints:

Past History

Birth History

Family History

Treatment History

EXAMINATION

General

Ocular Examination

Vision

Extra Ocular Movements

Anterior Segment

Evaluation of Squint

- Cover test
- Prism Bar Cover test
- Worth Four Dot test
- Pattern of Deviation

RETINOSCOPY

Subjective

Type of Amblyopia:

KEY TO MASTER CHART

S.No. – Serial number

M.R.D. No. Medical Records Department Number

Age – Age in years

Sex – M – Male

F – Female

R – Rural

U – Urban

RE – Refractive Error

H – Hypermetropia

M- Myopia

A – Astigmatism

V/A Visual Acuity

FIX – Fixation

U/B Unilateral / Bilateral

Laterality of AMB – Type of Amblyopia

T. AMB – Type - Type of Amblyopia

STR - STRABISMIC

ANT - ANISOMETROPIC

AME - AMETROPIC

VD - VISNN DEPRIVATION

M - MERIDIONAL

FIX - Type of Fixation

E – Eccentric

PEF - Perifoveal

PAF - Parafoveal

PER - Peripheral

NYS - NYSTAGMUS

No. 1 Absent

No. 2 Present

TYPS Type of Squint

No.1 ESO - ESOTROPIA

No.2 EXO - EXOTROPIA

No.3 HYP - HYPERTROPIA

No.4 HYPO- HYPOTROPIA

LBW - Low Birth Weight

No (1) Absent

No (2) Present

FHOS - Family history of squint

No. 1 absent

No. 2 Present

Master Chart

SL.	Name	MRD No.	Age	Sex	R/U	R.E.	V.A.	TYPA	FIX	LAT	NYS	TYPS	LBW	FHOS
1.	Jebakumar	483021	6	M	U	H	3/60NIP	STR	E	U	1	ESO	1	1
2.	Balaji	485016	5	M	U	M	6/36 WITH PH6/12	STR	PER	U	1	EXO	2	1
3.	Veeramani	413210	20	M	R	H	6/24	STR	PER	U	1	ESO ĉ TYPE	1	1
4.	Liyakath Ahamed	423160	3 ½	M	R	A	6/36	STR	PAR	U	1	EXO	2	2
5.	Jilani	397862	9	M	R	A	6/36	STR	PAR	U	1	ESO	1	1
6.	Silambarasman	421441	16	M	R	H	6/18	STR	PER	U	1	EXO	1	2
7.	Usha Rani	412987	48	F	R	M	6/24	ANI	PAR	U	1	-	1	1
8.	Vignesh	387921	6	M	R	M	6/24	ANI	PAR	U	1	-	1	1
9.	Roja	487321	6	F	U	A	6/60	STR	PAR	U	1	ESO	1	2
10.	Karthick	3876191	13	M	R	H	2/60	STR	E	U	1	ESOĉ HYPE	2	1

Master Chart

SL.	Name	MRD No.	Age	Sex	R/U	R.E.	V.A.	TYPA	FIX	LAT	NYS	TYPS	LBW	FHOS
11.	Mohammed Hajudeen	371622	4	M	R	H	6/24	STR	PAR	U	1	ESO	1	2
12.	Kokila	483217	11	F	R	M	6/24	AME	PER	B	1	EXO	1	1
13.	Vikram	421721	1	M	R	H	<6/60	STR	E	U	2	ESO	1	1
14.	Rahman	4036121	8	M	R	H	1/60	V.D	E	U	1	ESOĉ HYPO	1	1
15.	Nithya	4031261	3	F	U	H	6/36	STR	PAR	U	1	ESO	1	2
16.	Sivakumar	391621	8	M	U	H	6/12	STR	PER	U	1	EXO	1	2
17.	Manikam	391323	10	M	R	H	6/60	STR	E	U	1	ESO	1	1
18.	Sudha	391474	7	F	U	H	6/60	STR	PL	U	1	ESO	1	1
19.	Arumugam	487152	7	M	R	A	3/60	AME	PL	U	1	ESO	1	1
20.	Thenmozhi	497122	11	F	U	M	6/24	STR	PAR	U	1	EXD ĉ HYPER	1	1

Master Chart

SL.	Name	MRD No.	Age	Sex	R/U	R.E.	V.A.	TYPA	FIX	LAT	NYS	TYPS	LBW	FHOS
21.	Sumathy	491221	8	F	R	M	4/60	STR	PL	U	2	EXO	2	1
22.	Arumugam	392125	64	M	R	M	6/60	STR	PL	B	1	EXO	1	1
23.	Jenifer	392100	7	F	R	M	6/36	STR	PAR	U	1	EXO ĉ HYPER	1	1
24.	Jagadeesh	397166	3	M	R	H	6/36	STR	PAR	U	1	EXO	1	1
25.	Chitrangi	391800	9	F	R	H	6/24	STR	PAR	U	1	EXO	1	2
26.	Karthick	391722	9	M	R	H	2/60	STR	PL	U	1	EXO ĉ HYPER	1	1
27.	Prakash	399199	35	M	R	H	6/24	STR	PAR	U	1	EXO	1	1
28.	Joseph	392720	47	M	U	H	6/36	STR	PAR	U	1	ESO	1	1
29.	Balasubramanian	396726	4	M	R	H	1/60	STR	E	U	1	ESO	2	1
30.	Vigneshkumar	394220	8	M	R	M	6/18	STR	PAR	U	1	ESO ĉ HYPER	1	1

Master Chart

SL.	Name	MRD No.	Age	Sex	R/U	R.E.	V.A.	TYPA	FIX	LAT	NYS	TYPS	LBW	FHOS
31.	Gowri	392222	8	F	R	H	6/24	STR	PAR	U	1	ESO	1	1
32.	Naramadha	391233	2	F	R	H	<6/60	STR	E	U	2	ESO	1	2
33.	Rajkumar Pande	391166	11	M	R	H	6/60	V.D	E	U	1	EXO	1	1
34.	Jerald	392112	12	M	U	A	6/18	M	PER	U	1	-	1	1
35.	Kousalya	391231	6	F	R	H	6/36	V.D	PER	B	1	EXO	1	1
36.	Dhanush	391125	6	M	U	H	6/18	STR	FOV	U	1	ESO	1	2
37.	Ajithkumar	461205	8	M	R	A	6/18	STR	PER	U	1	ESO	1	1
38.	Selvi Soundarya	401075	12	F	U	M	6/18	STR	PER	U	1	EXO	1	1
39.	Mohan	401023	7	M	R	M	6/24	STR	PAR	U	1	EXO	1	2
40.	Tejaswari	401069	7	F	U	M	6/24	ANI	PAR	U	1	EXO	2	1

Master Chart

SL.	Name	MRD No.	Age	Sex	R/U	R.E.	V.A.	TYPA	FIX	LAT	NYS	TYPS	LBW	FHOS
41.	Vanisha	411233	9	F	R	H	6/24	STR	PER	U	1	ESO	1	2
42.	Janardhan	400111	5	M	R	A	6/36	STR	PAR	U	1	EXO ĉ HYPER	2	1
43.	Kameswaran	411332	3 ½	M	U	M	6/60	ANI	PL	U	1	ESO	1	1
44.	Sakthivel	401723	4	M	U	M	6/24	AME	PAR	U	1	EXO	1	1
45.	Manikandan	401222	11	M	R	M	6/36	STR	PAR	P	1	ESO ĉ HYPER	1	2
46.	Elumalai	401976	12	F	R	M	6/24	STR	PAR	U	1	EXO	1	1
47.	Bagyalakshmi	400111	5	F	R	H	1/60	VD	E	U	1	EXO	1	1
48.	Kamalesh	401224	6/12	M	U	M	< 6/60	ANI	E	U	2	ESO	1	1
49.	Bharathi	422105	7	F	U	M	6/24	AME	PAR	B	1	-	1	1
50.	Nagaraj	411122	28	M	R	H	5/60	STR	E	U	2	EXO	1	1

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LIST OF OPERATIONS PERFORMED

S.No	Name	Age / Sex	OP / IP No.	Date	Eye	Surgery
1	Vijayalakshmi	64/F	435225	20/07/2005	LE	ECCE with PCIOL
2	Arumugam	54/M	435296	27/07/2005	RE	ECCE with PCIOL
3	Geetha	28/F	431784	29/07/2005	LE	Incision and curettage
4	Venkatesan	34/M	431286	04/08/2005	RE	Excision
5	Visalakshi	68/F	432252	06/08/2005	RE	DCT
6	Nagamma	64/F	433358	08/08/2005	RE	ECCE with PCIOL
7	Dhanalakshmi	52/F	434552	10/08/2005	RE	I & D
8	Padmavathi	55/F	434689	02/09/2005	LE	Suturing of Corneal tear
9	Mohan	28/M	435002	09/09/2005	RE	Suturing of Lid tear
10	Munusamy	56/M	435100	16/09/2005	LE	ECCE with PCIOL
11	Virudammal	64/M	481154	23/09/2005	RE	ECCE with PCIOL
12	Prasath	18/M	481166	30/09/2005	RE	I & C
13	Vasantha	58/F	481177	02/10/2005	LE	DCT.
14	Kandasamy	68/M	471128	09/10/2005	LE	Evisceration
15	Maniammal	64/F	472228	16/10/2005	RE	Suturing of Corneal tear
16	Vedha	72/F	472215	23/09/2006	LE	DCT
17	Pappammal	50/F	472318	30/07/2006	LE	ECCE with PCIOL
18	Chinnappan	65/M	474812	04/08/2006	RE	Suturing of Corneal tear
19	Mahendran	60/M	474915	11/08/2006	RE	SICS with PCIOL
20	Manikkam	72/M	484501	18/08/2006	RE	SICS with PCIOL